

# Psychiatric Briefs

## The Moderating Effects of Coping Strategies on Major Depression in the General Population

Wang J, Patten SB

**Objectives:** This study evaluated the moderating effects of different coping strategies on the association between stressors and the prevalence of major depression in the general population. **Method:** The analysis included subjects from the Alberta (Canada) buy-in component (N = 1039) of the 1994–1995 National Population Health Survey (NPHS), each of whom were asked 8 questions regarding strategies for coping with unexpected stress arising from family problems and personal crises. The World Health Organization's Composite International Diagnostic Interview-Short Form for major depression was used to diagnose major depression. Logistic regression modeling was used to examine interactions between coping and life stress, thus allowing determination of the impacts of coping strategies in relation to psychological stressors on the prevalence of major depression. **Results:** There was no robust impact of coping strategies in relation to various categories of stress evaluated in the NPHS. Results suggested that the risk in women of major depression in the presence of financial stress and relationship stress (with a partner) was moderated by the use of the coping strategies "pray and seek religious help" and "talks to others about the situations." The risk of major depression in the presence of 1 or more recent life events, personal stress, relationship stress (with a partner), and environmental stress may be decreased in women by using emotional expression as a coping strategy. **Conclusion:** A differential impact on the prevalence of major depression in specific circumstances may be exhibited by different coping strategies. These findings may inform both prevention and treatment of depressive disorders.

(*Can J Psychiatry* 2002;47:167–173)

## From First Drug Use to Drug Dependence: Developmental Periods of Risk for Dependence Upon Marijuana, Cocaine, and Alcohol

Wagner FA, Anthony JC

**Background:** In focusing on the transition from drug use to drug dependence, this article presents new evidence on the risk for beginning to use marijuana, cocaine, and alcohol and the risks for progressing from initial use to dependence for each of these drugs. **Method:** Data from the National Comorbidity Survey, which included a representative sample of the U.S. population aged 15 to 54 years (N = 8098), were analyzed. Age- and time-specific risk estimates of beginning use of marijuana, cocaine, and alcohol and of becoming dependent on each drug, were acquired using survival analysis techniques. **Results:** Risk for initiating alcohol and marijuana use peaked at 18 years of age, approximately 2 years earlier than the peak risk for initiating cocaine use. Risk for meeting criteria for the clinical depen-

dence syndrome peaked from 17 to 18 years of age for individuals who used alcohol and marijuana and from 23 to 25 years of age for those who used cocaine. Cocaine dependence followed quickly from cocaine use; an estimated 5% to 6% of cocaine users became dependent on cocaine within a year of first use, and most individuals with cocaine dependence met criteria for dependence within 3 years after initial cocaine use. Although 15% to 16% of cocaine users had developed dependence within 10 years of first cocaine use, only about 8% of marijuana users and 12% to 13% of alcohol users developed dependence on those drugs within 10 years of initial use. **Conclusion:** This study found a noteworthy risk of developing cocaine dependence soon after initial cocaine use, with about 1 in 16 to 20 cocaine users becoming dependent on cocaine within the first year of cocaine use. The onset of the drug dependence syndrome is more insidious with alcohol and marijuana.

(*Neuropsychopharmacology* 2002;26:479–488)

## National Trends in the Outpatient Treatment of Depression

Olfson M, Marcus SC, Druss B, et al.

**Background:** Depressive disorders are highly prevalent in the United States. Because of changing health care environments and recent advances in pharmacotherapy, increased attention has been paid to trends in outpatient treatment of depression. This study compared trends in outpatient treatment of depression in 1987 and 1997. **Method:** Service utilization data from 2 nationally representative surveys of the U.S. general population—the 1987 National Medical Expenditure Survey (N = 34,459) and the 1997 Medical Expenditure Panel Survey (N = 32,636)—were compared. Participants who made 1 or more outpatient visits owing to DSM-IV major depressive disorder (single episode or recurrent), dysthymic disorder, or depressive disorder not otherwise specified were defined as having received treatment for depression. Main outcome measures included rate of treatment, use of psychotropic medication, psychotherapy, number of outpatient treatment visits, type of health care professional, and payment source. **Results:** There was an increase in the rate of outpatient treatment for depression from 1987 (0.73 per 100 persons) to 1997 (2.33 per 100 persons;  $p < .001$ ). Of those treated, the proportion who received antidepressant medications increased from 37.3% in 1987 to 74.5% in 1997 ( $p < .001$ ), while the proportion who received psychotherapy declined (71.1% in 1987 vs. 60.2% in 1997;  $p = .006$ ). A decline was found in the mean number of depression treatment visits per year, from 12.6 to 8.7 ( $p = .05$ ). The proportion of patients treated by physicians increased (68.9% vs. 87.3%;  $p < .001$ ), and treatment costs were increasingly paid by third-party payers (39.3% vs. 55.2%;  $p < .001$ ). **Conclusions:** The proportion of the population receiving outpatient treatment for depression increased markedly between 1987 and 1997. Greater involvement

of physicians, increased use of psychotropic medications, and greater availability of third-party payment, combined with fewer outpatient visits and less use of psychotherapy, characterized this time period. These changes were contemporaneous with the introduction of better-tolerated antidepressant medications, increased penetration of managed care, and the development of quick and efficient procedures for diagnosing depressive disorders in clinical practice.

(*JAMA* 2002;287:203–209)

### Mortality Associated With Sleep Duration and Insomnia

Kripke DF, Garfinkel L, Wingard DL, et al.

**Background:** Complaints about insufficient sleep or chronic insomnia are often made by patients who believe that they need 8 hours of sleep per night. The sleep durations that predict optimal survival, and whether insomnia signals mortality risks, may guide treatment strategies. **Method:** Data were taken from the 1982 Cancer Prevention Study II of the American Cancer Society, in which participants were asked about their duration of sleep and frequency of insomnia. Whether an association existed between sleep duration or frequency of insomnia on the one hand and excess mortality up to 1988 on the other was determined using Cox proportional hazards survival models that controlled for demographics, habits, health factors, and use of various medications. **Results:** The study population comprised more than 1.1 million men and women from 30 to 102 years of age. Individuals who slept 7 hours per night had the best survival. Significantly increased mortality hazard was experienced by those who slept 8 hours or more or 6 hours or less per night. The increased risk was greater than 15% for those reporting more than 8.5 hours or less than 3.5 to 4.5 hours of sleep per night. Reports of "insomnia," however, were not associated with excess mortality hazard. After control for reported sleep durations and insomnia, use of prescription sleeping pills was associated with significantly increased mortality. **Conclusions:** Little risk distinct from comorbidities seems to be associated with short sleep duration and insomnia. Although slight risks were associated in this study with sleep duration greater than 8 hours and sleeping pill use, causality remains unproven and these risks need further study.

(*Arch Gen Psychiatry* 2002;59:131–136)

### Psychological Sequelae of the September 11 Terrorist Attacks in New York City

Galea S, Ahern J, Resnick H, et al.

**Background:** The terrorist attacks of September 11, 2001, were unprecedented in scope in the United States. In this study, the prevalence and correlates of acute posttraumatic stress disorder (PTSD) were assessed in residents of Manhattan 5 to 8 weeks after the attacks. **Method:** A representative sample of adults living south of 110th street in Manhattan (the part of Manhattan closest to the World Trade Center) was contacted for interview by telephone via random-digit dialing. The interview included questions about demographic characteristics, exposure to the events of September 11, and psychological symptoms after the

attacks. PTSD was assessed using the PTSD questionnaire from the National Women's Study, and major depressive disorder, using a modified, validated version of the Structured Clinical Interview for DSM-IV. **Results:** A total of 7.5% of the study sample (N = 1008) reported symptoms consistent with a current (i.e., within the previous 30 days) PTSD diagnosis related to the attacks, and 9.7% reported symptoms consistent with a current depression diagnosis. The prevalence of PTSD was 20.0% among those living in the immediate vicinity of the World Trade Center (i.e., south of Canal Street). In a multivariate model, predictors of PTSD were Hispanic ethnicity, 2 or more prior stressors, a panic attack during or soon after the events, residence south of Canal Street, and loss of possessions due to the events. Depression was predicted by Hispanic ethnicity, 2 or more prior stressors, a panic attack, a low level of social support, the death of a relative or friend during the attacks, and loss of a job due to the attacks. **Conclusions:** The attacks of September 11 were followed by a substantial burden of acute PTSD and depression in Manhattan. Current PTSD was predicted by experiences involving exposure to the attacks, and current depression was predicted by losses resulting from the events. Substantial psychological morbidity in the population may be found in the aftermath of terrorist attacks.

(*N Engl J Med* 2002;346:982–987)

### Reboxetine, a Selective Norepinephrine Reuptake Inhibitor, Is an Effective and Well-Tolerated Treatment for Panic Disorder

Versiani M, Cassano G, Perugi G, et al.

**Background:** Tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRIs) as well as benzodiazepines have been shown to be effective for the treatment of panic disorder. The introduction of SSRIs has enabled a greater understanding of the role of serotonin in the etiology of panic disorder; however, the role of norepinephrine has been more challenging to ascertain. The aim of this study was to determine the efficacy and tolerability of reboxetine, a novel selective norepinephrine reuptake inhibitor, in patients with panic disorder with and without agoraphobia. **Method:** Eighty-two patients (aged 18–65 years) with DSM-III-R panic disorder, with or without agoraphobia, were randomly assigned to receive 6 to 8 mg/day of reboxetine (42 patients) or placebo (40 patients) for 8 weeks in this placebo-controlled, parallel-group, double-blind clinical trial. **Results:** Of the 82 patients enrolled in the trial, 75 were considered in the analysis (37 patients in the reboxetine group and 38 patients in the placebo group). At last assessment, there was a significant reduction in the mean number of panic attacks (range, 9.3–1.2) and phobic symptoms (range, 8.1–3.2) in the reboxetine group compared with the placebo group (ranges, 8.5–5.8 and 7.7–5.2, respectively;  $p < .05$ ). Improvement in Hamilton Rating Scale for Depression, Hopkins Symptom Checklist-90, and Sheehan Disability Scale scores were also greater in the reboxetine group compared with the placebo group. Adverse events reported more frequently with reboxetine than placebo included dry mouth (36% vs. 16%), constipation (27% vs. 22%), and insomnia (26% vs. 22%). **Conclusion:** Reboxetine was effective and well tolerated in the treatment of panic disorder.

(*J Clin Psychiatry* 2002;63:31–37)